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SECONDARY POISONING HAZARDS ASSOCIATED WITH RODENTICIDE USE

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ABSTRACT

Relatively few field studies have been conducted to evaluate secondary hazards to wildlife from rodenticide use. In the United States (U.S.), field studies of rodenticide hazards have included both acute and chronic compounds. Techniques employed in these studies have included carcass counts, direct counts, indirect counts, nest site monitoring, radiotelemetry, habitat and diet evaluation, necropsy, and residue analysis. Results have been interpreted using data on exposure (i.e., use of treated areas, acreage treated within a home range), survival/mortality, residue chemistry, and necropsy.

Among acute compounds, zinc phosphide generally is not secondarily hazardous, while strychnine can pose a secondary hazard if predators consume stomach or cheek-pouch contents of poisoned prey. Although 1080 (sodium monofluoroacetate) can pose a secondary hazard to mammalian predators, the risk to raptors is minimal. Anticoagulants potentially are secondarily hazardous; they can pose a substantial hazard to raptors. Hazards associated with any one rodenticide may vary significantly depending upon use pattern (e.g., commensal vs. field) and the foraging behavior and habitat use of nontarget wildlife. Environmental concerns over rodenticide hazards to wildlife are increasing greatly and will affect future U.S. registrations.

INTRODUCTION

The hazards to wildlife associated with rodenticide use have received relatively limited attention. In the U.S., nontarget hazards are addressed by the U.S. Environmental Protection Agency (EPA) and rodenticide manufacturers as part of the current registration process. Hazard concerns are reflected in restrictions on rodenticide labels.

Although some laboratory research has been conducted to help assess poisoning hazards from rodenticide use, these studies usually have involved a limited number of species and highly controlled conditions. More importantly, few field studies have been conducted to evaluate the specific hazards to nontarget wildlife from the various use patterns, active ingredients, and formulations of rodent bait available today.

In the U.S., more emphasis now is being placed on the evaluation of nontarget hazards from rodenticide use. Of the field studies that have been conducted, most were directed by the U.S. Fish and Wildlife Service. The objective of this paper is to review some of those field studies and, in particular, to discuss methodologies, the secondary hazards to predators associated with various compounds and use patterns, and aspects of hazard evaluation and interpretation.

METHODS

Primary poisoning involves consumption of rodenticide bait by a nontarget animal (such as a grain-eating bird or mammal), while secondary poisoning results when a predator or scavenger consumes a target or nontarget animal that has consumed a rodenticide bait. Even when evaluating only secondary hazards, there is an inherent need to consider primary poisoning of nontarget wildlife, since this can provide an array of potential food-chain links to the toxicant for predators.

Most field studies have focused on either primary or secondary hazards and a limited number of nontarget species. Logistics, equipment, and capture limitations often preclude broader efforts to evaluate simultaneously both types of hazards or to monitor numerous species. These limitations are particularly apparent when studying predators (secondary hazards).

Carcass searches have been used to assess hazards to nontarget wildlife (Hegdal et al., 1986), but these provide limited data. Infrequently is any animal found dead in the wild because of cryptic coloration, vegetative cover, and mortality occurring in hidden locations such as den sites. Also, scavengers may quickly consume carcasses (Balcomb, 1986). Additionally, because of the delayed action of some rodenticides and the ranging ability of some nontarget wildlife (e.g., predators), mortality may occur at a considerable distance from a treated area. Carcass searches also have been used to find target animals, to determine their residue loadings. Thus, potential secondary hazards to predators have been extrapolated (Barnes et al., 1985).

Direct counts of individuals present before and after treatment demonstrate the persistence or reduction of individuals. Such counts have been used successfully in primary hazard assessment by marking birds, noting their territories, and monitoring their presence pre- and post-treatment (Hegdal and Gatz, 1976). However, direct counts do not provide adequate data on many predatory species, especially nocturnal ones, because of their secretive nature. For this reason, direct counts may be more useful in primary hazard evaluations because of the observability of the species at potential risk and their numerical abundance.

Indirect counts also have been used as an index to numerical changes in nontarget populations. These techniques can include track counts and call census (Hegdal et al., 1986). However, as with direct counts (especially of unmarked individuals), these techniques do not document the actual mortality of individuals; and the results can be distorted by population changes occurring independent of rodenticide use (such as from weather effects or emigration).

Monitoring of nest or den sites and determining success have been important and useful components of secondary hazard evaluations (Hegdal et al., 1986). Not only might adult mortality be determined, but also that of young, and thus, both long- and short-term impact on productivity. Artificial nest sites have been used in some studies to provide specific locations to look for nesting raptors and to monitor nesting success (Hegdal and Blaskiewicz, 1984).

Radiotelemetry has been the technique used most frequently in the U.S. to evaluate secondary poisoning hazards (Merson et al., 1984; Hegdal et al., 1986). This method provides specific information on the fate of individuals post-treatment. It also provides critical information on habitat use by nontarget animals; such data can be essential in interpreting the presence or absence of a potential hazard. Additionally, radiotelemetry allows for carcasses to be located for necropsy and residue analysis. Although radiotelemetry is an outstanding tool for detecting hazards, it is an expensive undertaking in time and equipment and requires skilled personnel and the ability to capture animals to radio-equip. However, use of telemetry is important in evaluating secondary hazards, since predators frequently are difficult to observe or locate.

Necropsy and residue analyses have provided strong supportive evidence for documenting the most probable cause of death when mortalities have been observed. However, necropsy and residue information alone may not provide conclusive evidence for establishing cause of death, but simply factors symptomatic of rodenticide-poisoning. Residue loads may not be closely correlated with mortality (Hegdal and Colvin, 1988). Additionally, in some situations (such as with 1080), poisoning symptoms may not be obvious or residue detection readily achievable (Hegdal et al., 1981).

Field studies of rodenticide hazards typically are not exclusive in the techniques used. Information assembled from population evaluation, behavioral studies of habitat use and diet, necropsy, and residue analyses all may be essential for implicating rodenticide poisoning as a mortality factor.

RESULTS AND DISCUSSION

Field evaluations of rodenticide hazards have included studies in the U.S. on three acute compounds: zinc phosphide, strychnine, and 1080 and also the anticoagulants diphacinone and brodifacoum. Additional information on the toxicity of these compounds and potential hazards has been acquired through laboratory studies.

Zinc Phosphide

This compound can pose primary hazards to rabbits, gallinaceous birds, waterfowl, and seed-eating birds (Rudd and Genelly, 1956; Janda and Bosseova, 1970; Hegdal and Gatz, 1977a). However, it is generally not secondarily hazardous. When used for vole control in orchards, Hegdal and Gatz (1977a) did not observe adverse effects on mammalian or avian predators. In another orchard study, Hegdal and Colvin (1988) also did not observe a relationship between exposure to zinc phosphide and raptor mortality.

Laboratory studies repeatedly have demonstrated a lack of secondary toxicity to both birds and mammals from zinc phosphide (Brock, 1965; Siegfried, 1968; Bell and Dimmick, 1975; Schistoskey, 1975). For example, Evans et al (1970) reported that golden eagles (*Aquila chrysaetos*), great horned owls (*Bubo virginianus*), and coyotes (*Canis latrans*) which received multiple feedings of zinc phosphide killed black-tailed jackrabbits (*Lepus californicus*) and showed no symptoms of secondary intoxication. Steininger (1952) concluded that zinc phosphide is so extensively decomposed in the digestive tract of poisoned rodents that it cannot subsequently injure rodent-eating birds.

Strychnine

Field studies have been conducted by Hegdal and Gatz (1976, 1977b), Fagerstone et al. (1980), and Anthony et al. (1984) to evaluate potential hazards from using strychnine baits for rodent control. Each study included grain bait containing 0.5 percent strychnine. However, the first study (1976) focused on underground baiting for plains pocket gopher (*Geomys bursarius*) in old-fields using a burrow-builder; the second (1977b) involved surface baiting for Richardson's ground squirrel (*Spermophilus richardsonii*) in rangeland; and the third (1980) and fourth (1984) dealt with hand-baiting of underground burrows for western pocket gophers (*Thomomys mazama*) in conifer plantations.

Hegdal and Gatz (1976) regarded underground baiting with strychnine as relatively safe for nontarget wildlife. In contrast, Hegdal and Gatz (1977b) observed a significant hazard to seed-eating birds from surface baiting. In the plantation studies, Fagerstone et al. (1980) observed some primary poisoning of small mammals, while Anthony et al. (1984) found that nontarget populations of golden-mantle ground squirrels (*Spermophilus lateralis*) were reduced by 50 to 70 percent post-treatment.

Secondary poisoning was not observed during either of the two strychnine studies by Hegdal and Gatz (1976, 1977b), even though numerous raptors, mammalian predators, and nest sites were monitored. Hegdal et al. (1981), and also Anthony et al. (1984), described the secondary hazard from strychnine as minimal, although they believed that secondary poisoning could occur if the stomach or cheek-pouch contents of poisoned prey were consumed. Schitoskey (1975) demonstrated secondary strychnine poisoning of kit fox (*Vulpes macrotis*) in the laboratory, while Marsh et al. (1987) noted that coyotes (*Canis latrans*) tended to reject portions of gastro-intestinal tracts of poisoned squirrels, resulting in a minimal secondary hazard.

1080

Compound 1080 was evaluated by Hegdal et al. (1986) for primary and secondary hazards when used to control California ground squirrels (*Spermophilus beecheyi*) in rangeland. They found that primary hazards existed for nontarget rodents and rabbits, but there were few incidences of primary poisoning of seed-eating birds.

Hegdal et al. (1986) found that canids and felids were at most risk from secondary 1080 poisoning but that the risk to predatory birds was low. They also reported some mortality of insectivorous birds that apparently fed on ants killed by 1080. The secondary toxicity of 1080 to coyotes also was recently demonstrated by Marsh et al. (1987) in the laboratory.

Marsh et al. (1987) commented that the secondary hazards with 1080 may be reduced in the field by lowering bait concentrations and the amount of bait applied. Recent investigations (Matschke and Hegdal, 1985) indicate that 1080 efficacy can be maintained, at least for some species, at lower bait concentrations.

First-generation Anticoagulants

Currently, only diphacinone and chlorophacinone are registered and used for field rodent control, such as for microtine rodents in orchards. Field data on hazards associated with first-generation anticoagulants are limited. However, some secondary hazard data are available from laboratory studies (Evans and Ward, 1967; Mendenhall and Pank, 1980; Townsend et al., 1981).

Hegdal (1985) evaluated the primary hazard to game birds from diphacinone when used to control microtine rodents in apple orchards. He found the primary hazards to these gallinaceous birds to be low, even though many fed extensively on the bait. Secondary hazards were not evaluated, but observations of primary poisoning suggested a potential hazard to predators, including man.

Second-generation Anticoagulants

Hazards to wildlife associated with anticoagulant rodenticides have been discussed by Kaukeinen (1982), and two major field studies have been conducted to evaluate secondary hazards.

In a study of Norway rat (*Rattus norvegicus*) and house mouse (*Mus musculus*) control on farmsteads (in and around buildings), Hegdal and Blaskiewicz (1984) and Colvin (1984) found that the hazard to barn owls (*Tyto alba*) from brodifacoum bait (50 ppm) was low. Although the owls nested and roosted on farmsteads, they demonstrated selective behavior for grassland foraging habitat and microtine rodents. Neither the location of rodenticide use nor the target species were frequently used by owls as a foraging resource.

In contrast to the barn owl study, Hegdal and Colvin (1988) found that when brodifacoum bait (10 ppm) was evaluated for control of microtine rodents in orchards, there was a substantial hazard to eastern screech-owls (*Otus asio*). This study demonstrated the poisoning hazard that can be associated with second-generation compounds and the impact when the target species is the same for both rodenticide and predator.

No second-generation anticoagulant currently is registered in the U.S. for non-commensal (field) uses. Hazards to nontarget wildlife will probably limit such registrations.

Field studies of rodenticide hazards provide data on nontarget wildlife in settings that allow for normal display of behavioral characteristics, the effects of ecological conditions, and typical use patterns of rodenticide bait. However, field evaluations are expensive; can involve numerous personnel; are difficult to coordinate on a large scale; and can encounter problems related to weather, animal capture, and equipment maintenance in the field. Additionally, given a host of environmental factors that can affect population change, it may be difficult to distinguish rodenticide-related effects from those resulting from other environmental or behavioral factors.

Laboratory studies, in contrast, involve a highly controlled situation. However, the behavioral patterns of wildlife in captivity may be altered, and limited space and stress may contribute to poisoning symptoms (Jaques and Hiebert, 1972). Importantly, rodenticide exposure is actively and rigidly controlled by the experimenter in the laboratory, rather than being a passive encounter during normal feeding and habitat use, as occurs in the field. Because specific use patterns and formulations of bait, relative to foraging behavior of the nontarget animal, cannot be taken easily into direct account, dose levels administered in the laboratory become tests of toxicity rather than an absolute determination of hazard.

Laboratory studies can generate important base-line information for determining whether a field study should be initiated and also the species that may be at greatest risk (avian or mammalian). If a rodenticide is demonstrated in the laboratory not to be hazardous to nontarget wildlife or to certain nontarget species, field studies to test such questions may not be necessary or of critical importance. Conversely, if primary or secondary toxicity is demonstrated readily in the laboratory, decisions then can be made to either proceed with a field study or not to pursue rodenticide registration or certain use patterns.

Hazard studies can be viewed sequentially, starting with lab studies, short-term field studies, and then long-term field studies (Colvin and Hegdal, 1988). At each plateau, information obtained may signal the need to proceed with additional research or be adequate to demonstrate or project the potential hazard. Credible decisions and interpretations must be made at each step, since it simply is not possible to test every conceivable species in the laboratory or to monitor numerous species in prolonged field studies.

Field studies readily can become simply a test of survival or mortality. Of critical importance, however, are the reasons why survival or mortality were observed post-treatment. For example, interpretation of results and the predictability of hazard to individuals require information on nontarget species foraging behavior and habitat use (Colvin, 1984); the acquisition of such information needs to be given greater emphasis in field studies. An ecological approach is demanded, rather than simply asking, "How many lived and how many died?"

Similarly, sound ecological information on nontarget species is important for explaining changes in nontarget populations. Otherwise, the mere presence of a rodenticide in the environment and a reduced nontarget population can be falsely related. For example, the drastic decline of barn owl populations in the midwestern United States over the past 30 years was attributed to rodenticide use on farmsteads. However, research on population dynamics and habitat requirements showed that loss of grassland foraging habitat was the principal factor in species loss, not farmstead use of rodenticide (Colvin, 1985).

The hazards associated with a particular rodenticide may be influenced by the formulation (treated grain, pellets, wax blocks), concentration of active ingredient in bait, use pattern, and target species. In fact, each of these factors may drastically alter the potential hazard. For example, studies of brodifacoum use on farmsteads (for commensal rodent control) and in orchards (for vole control) have illustrated how the same active ingredient can pose a minimal or substantial hazard, depending upon the use pattern and target species.

There is growing concern in the U.S. regarding the hazards associated with rodenticide use. Concern over the hazard to raptors (at the apex of food pyramids) is particularly acute. Because rodenticides used to control field rodents (e.g., voles, pocket gophers, ground squirrels) pose the most direct exposure to wildlife, particularly predators, those compounds and use patterns will receive greater scrutiny. We anticipate that for rodenticides in use, but for which there are limited data available on nontarget hazards, additional data will be required by EPA to maintain registrations. Also, nontarget hazard data will continue to be a key registration requirement for proposed rodenticides and use patterns.

Development of rodenticides with low toxicity to avians and low secondary toxicity should be given particular attention, and these will have the greatest opportunity for registration for field rodent control. Reduction in treatment rates, lower concentration of active ingredient in bait, and restricted use patterns also may receive greater emphasis. Environmental concerns dictate that we, as vertebrate pest researchers and managers, provide effective, yet environmentally responsible, control measures.

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